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Bacterial Endocarditis in a Patient with a Saphenous Vein Graft A-V Fistula Receiving Dental Work

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SURGICALLY CREATED arteriovenous (AV) fistulae in dogs have resulted in spontaneous bacterial endocarditis about four to six weeks afterward.[1] This model suggests that patients with AV fistulae receiving hemodialysis could be a high risk group for the occurrence of bacterial endocarditis. Recently reports of bacterial endocarditis associated with the use of subcutaneous AV fistulae in hemodialysis patients have appeared in the literature.[2] We present here another case of bacterial endocarditis associated with the use of a saphenous vein graft AV fistula for hemodialysis.

Report of a Case

The patient, a 37-year-old black man without previous valvular heart disease and with chronic renal failure, entered the hospital in florid congestive heart failure, five months after having begun chronic hemodialysis. He had initially received dialysis three times per week via a Scribner (Teflon-Silastic) AV shunt placed in the right forearm, but when this clotted four months later, a subcutaneous, saphenous vein graft AV fistula was surgically created in the left forearm and the sterile Scribner shunt was removed. At about this time, the patient also began regular visits to his dentist for scaling and filling of carious teeth. Two weeks later, the patient noted sudden onset of fever, chills, weakness and malaise. A false aneurysm in the AV fistula was drained and tetracycline was prescribed, 250 mg a day by mouth. Cultures of specimens from the aneurysm site grew enterococci, while blood and urine cultures at that time were sterile. The fistula healed, and dialysis was continued through this route. Five days before admission the diastolic blood pressure had fallen to 40 mm of mercury. Two days before admission, results of cardiac examination were considered normal.

Past history was of note in that the patient had had a previous anaphylactoid reaction to penicillin.

On admission the patient was acutely ill, with temperature of 38°C (100.4°F), blood pressure of 140/40 and regular pulse at 120 beats a minute. No peripheral stigmata of bacterial endocarditis were present. The patient was in frank biventricular congestive heart failure with a hyperactive precordium and a grade 3/6 systolic ejection murmur at the aortic area radiating into the carotid arteries, as well as a grade 3/6 diastolic murmur heard at the aortic area. Peripheral signs of aortic insufficiency were present. The AV fistula was clinically free of infection.

The patient was given one 80 mg dose of gentamicin intramuscularly and cephalosporin, 2 grams intravenously every eight hours, pending the result of blood cultures, which one day after admission revealed enterococci. The other drugs were discontinued, and vancomycin was given, 1 gram in one dose intravenously. Cultures of two urine specimens, obtained before treatment, were sterile. Despite rapid digitalization, falling diastolic blood pressure and increasing congestive heart failure culminated in cardiac arrest on the third hospital day.

Postmortem examination revealed left ventricular hypertrophy and extensive vegetations on the aortic valve. There was a rent in the noncoronary cusp extending from the base of the cusp to the free margin. The left coronary cusp was also perforated. No inflammatory changes were seen in the genitourinary tract. The left renal artery was 80 percent occluded by a large arteriosclerotic plaque, and the renal parenchyma was that of endstage kidneys with arterial changes consistent with benign nephrosclerosis, but no evidence of pyelonephritis. The AV fistula was not examined histologically.

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Discussion

In this case of acute bacterial endocarditis involving a previously normal aortic valve occurring one month after commencing hemodialysis via a subcutaneous AV fistula, it is possible that bacteremia occurred following repeated needle puncture of the fistula during the course of dialysis. However, the type of organism isolated (enterococci) suggests a different source. There was no clinical, bacteriologic or pathologic evidence of urinary tract infection, and the patient had not undergone urological instrumentation before admission. Thus, the genitourinary tract, the most common portal of entry in enterococcal endocarditis, was not the source in this case. On the other hand, the patient underwent dental procedures during the month before the onset of his illness, and resultant bacteremia of enterococci may have been the basis of endocarditis. Although this is an uncommon portal of entry for enterococci, it is important to note that both patients reported by Goodman et al[2] who had endocarditis following dialysis through an AV fistula, had undergone dental procedures just before the onset of acute illness. Moreover, in one of these patients enterococcus was the responsible organism; culture of the other patient's blood grew out S. viridans. In the first patient, the clinical course—including the absence of preexisting valvular disease, acute aortic insufficiency and fulminant congestive failure—was very similar to that in the case we have described.

The pathophysiology of bacterial endocarditis in the presence of an AV fistula without preexisting valvular damage is not well defined, although a number of hypotheses have been entertained.[1,5] One such idea has been that the increase in cardiac output occurring as a consequence of AV shunting results in trauma to the aortic valvular cusps and in an anatomical deformity which increases the susceptibility to growth of organisms on the valve following transient bacteremia. Lillehei et al[1] showed that an AV fistula of greater than 15 mm diameter surgically created in dogs resulted in spontaneous bacterial endocarditis about four to six weeks after creation of the shunt. A clinical correlation comes from the report by Cutler et al,[4] who described a patient with aortic valvular damage and bacterial endocarditis occurring as a result of traumatic creation of an AV fistula.

The susceptibility of the fistula itself to form a nidus of infection for seeding of bacteria is suggested by the work of Rodbard,[5] who noted that hemodynamically the flow from a high into a low pressure sink as well as alteration in laminar flow through this area may play a role in infection at this site. Possibly certain altered host defenses found in renal failure[6] contribute to the pathogenesis of endocarditis in this setting.

While the relationship between AV fistulae and bacterial endocarditis has not been definitely demonstrated in humans, from the increasing numbers of cases reported to date, it seems advisable that patients with such fistulae should receive prophylactic antibiotics while undergoing dental procedures. The choice of drugs should be directed against both vividans and enterococcus groups of Streptococci. Thus a combined penicillin and streptomycin regimen, or vancomycin in patients with hypersensitivity to these agents, should be recommended. In light of the underlying renal failure, dosage should be appropriately adjusted. In the case of vancomycin, the fact that this antibiotic is non-hemodialyzable must be taken into consideration.

Summary

Case reports of septicemia, septic pulmonary embolism and bacterial endocarditis have been associated with the use of subcutaneous arteriovenous (AV) fistulae in hemodialysis patients. In the case, here presented, of bacterial endocarditis occurring in a patient undergoing chronic hemodialysis with a subcutaneous AV fistula, the patient was undergoing dental procedures in the weeks before the onset of infection. This case and two other previously reported cases suggest that patients with AV fistulae undergoing dental procedures should receive prophylactic antibiotics whose spectrum includes coverage against enterococci.

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